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Issue Date: 03 December 2002

Case No. 1999-BLA-00180

In the Matter of:

CHARLES E. BAIRD,
Claimant

v.

KEY MINING, INCORPORATED.,
KLINE COAL COMPANY, INC.,
Employer

AMERICAN MINING INSURANCE
COMPANY,
Carrier

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest

DECISION AND ORDER ON REMAND

This matter is before me on remand from the Benefits Review Board following my award of benefits to the Claimant, Charles Baird, pursuant to the provisions of Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977 (hereinafter referred to as "the Act"), 30 U.S.C. § 901 *et seq.*, and the regulations issued thereunder at Title 20 of the Code of Federal Regulations ("C.F.R."). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a chronic dust disease of the lungs arising from coal mine employment and is commonly known as black lung.

The Employer has stipulated that Mr. Baird has coal workers' pneumoconiosis and is totally disabled (Tr. 10). In my decision and order of April 20, 2000, I found that Claimant's total disability was due, at least in part, to coal workers' pneumoconiosis. I reached my determination, giving full credit to the opinion of his treating physician, Dr. Parrish, that both coal workers' pneumoconiosis and cigarette smoking were contributing factors to the miner's disability. Dr. Parrish's diagnosis of complicated pneumoconiosis was not credited, and Claimant was found not to be entitled to invocation of the irrebutable presumption of total disability due to coal workers' pneumoconiosis under section 718.304. This latter determination was based on a finding that Dr. Parrish's diagnosis of complicated pneumoconiosis was not documented by X-ray

or CT- Scan reports in the record before me, and thus was outweighed by contrary probative evidence. (D & O Awarding Benefits, dated April 30, 2000, at p. 15) *See Hoffman v. B & G Construction Co.*, 8 BLR 1-65 (1985); *Hess v. Clinchfield Coal Co.*, 7 BLR 1-295 (1984).

In its appeal before the Benefits Review Board ("the Board"), the Employer argued that I did not adequately explain why I discounted the opinions of eight physicians (Drs. Abdul Dahhan, Gregory Fino, WKC Morgan, Ben Branscomb, Lawrence Repsher, Emory Lane, Bruce Broudy and L. J. Seargeant) whom it contends found the Claimant's total disability to be unrelated to coal workers' pneumoconiosis. The Board agreed and remanded for a re-evaluation of the weight to be accorded the relevant physician opinions, and reconsideration of the issue of whether pneumoconiosis is a "substantially contributing cause" of the Claimant's total disability.

The Parties have been accorded an opportunity to submit briefs on remand. Employer submitted its brief . Claimant, pro se, responded by submitting an update of his medical condition prepared by his treating physician, Dr. Richard Parrish. In his report, Dr. Parrish included office notes and x-ray reports. He reported that the miner's ventilatory tests in 2000 showed a deterioration in his pulmonary function, and that an x-ray taken in September of 2000 showed lung changes which had progressed to large conglomerate lesions in both upper lung fields, Category C, 3. He diagnosed complicated pneumoconiosis with progressive radiographic changes and a progressive decline in pulmonary function. He opined that the miner's pulmonary impairment is due, in large part, to his complicated coal workers' pneumoconiosis which arises from his coal mine employment. Dr. Parrish's most recent opinion is appended hereto as Attachment I.

Claimant's submission of this new evidence will be considered an informal motion to reopen the record. As such, the request is denied for the following reasons. The Board's remand decision directs that I re-examine the record evidence before me at the time I rendered my previous decision. The evidence submitted by the Claimant in response to my briefing order was generated after that date. While an administrative law judge has broad discretion in resolving procedural issues, absent a compelling reason, she is not required to reopen the record on remand from the Board unless there has been a change in the legal standard which was in effect at the time of the hearing. See *Cal-Glo Coal Company v. Yeager*, 104 F. 3d 827 (6th Cir. 1997). *Harlan Bell Coal Co v. Lemar,* 904 F. 2d 1042, 14 BLR 2-1 (6th Cir. 1990). In the instant case, there has been no supervening change in the legal standard applicable to the issues raised herein which would necessitate reopening the record.

In the Sixth Circuit, the jurisdiction in which this claim arises, the U.S. Court of appeals has adopted the "contributing cause" standard, which was in effect at the time I rendered my prior decision. *Adams v. Director, OWCP*, 886 F. 2d 818 (1989); *Peabody Coal Co. v. Smith*, 127 F. 3d 504, 507 (6th Cir. 1997). See also, *Scott v. Mason Coal Co.*, 14 BLR 1-37 (1990)(en banc). Under that standard, the Claimant must prove that his totally disabling respiratory or pulmonary impairment is due

"at least in part" to his pneumoconiosis. The new regulations promulgated by the Department of Labor pursuant to the Act, which became effective on January 19, 2001, articulate a "substantial contributing standard". This standard requires that pneumoconiosis have either a "material adverse effect" on the miner's respiratory pulmonary condition or" materially worsen" his totally disabling respiratory or pulmonary impairment caused by a disease or impairment unrelated to coal dust exposure. *§718.204(c)(1)(i) and (ii) (2000).* The revision in the regulations adds the term "material" to reflect the view that evidence that pneumoconiosis makes only a negligible inconsequential or insignificant contribution to disability is insufficient to establish that pneumoconiosis is a substantially contributing cause. *See 65 Fed Reg. 79, 946 (12/20/2000).* The revised regulations do not change the "contributing cause" standard. Rather, the regulatory revisions incorporate existing case law in *Peabody Coal, supra.,* which defines the "contributing cause standard" to mean that the miner's pneumoconiosis must be more than "de minimus" or infinitesimal factor in the miner's total disability. Since there has not been a significant change in the legal standard relating to the causation of total disability under the applicable regulations, I find no "good cause" to reopen the record, and Claimant's motion will be denied. *See White v. Director, OWCP,* 7 BLR 1-348 (1984). If following this decision, Claimant wishes to have his claim reevaluated based on this new evidence, a request for modification should be submitted to the District Director, Office of Workers' Compensation Programs.

Issue

The sole issue on remand is whether the miner's impairment is caused by coal workers' pneumoconiosis. The physicians' opinions are summarized in detail in my prior decision. *See Decision and Order Awarding Benefits, 1999 BLA 00180, April 30, 2000* at pp. 7-17. In weighing the opinions of the physicians, who addressed the cause of the miner's total disability, I make the following findings.

Relevant Medical Opinion Evidence[1]

Claimant's treating physician, Dr. Richard Parrish has treated the Claimant since 1994, and has consistently diagnosed coal workers' pneumoconiosis by chest x-ray and CT -Scan, and chronic obstructive pulmonary disease. In his disability assessments, he has noted a disabling and worsening severe obstructive lung disease related to the miner's coal mine work experience and his cigarette smoking. He was unable to find either chronic obstructive pulmonary disease or coal workers' pneumoconiosis exclusively responsible for the development of the miner's obstructive lung disease. But, he opined that given his occupational hisotry, chest x-rays, pulmonary function studies and smoking history, the miner's coal mine employment and his coal workers' pneumoconiosis are contributing factors to his disability. (See eg. reports of Dr. Parrish dated May 23, 1994, November 10, 1994, and July 13, 1995; DX 28, DX 33)

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[1] The medical reports of record were summarized in detail in my prior decision are incorporated herein. *See Decision and Order Awarding Benefits,* 4/30/2000, at pp. 7-17.

Dr. L. J. Seargeant diagnosed moderately severe chronic obstructive pulmonary disease and pneumoconiosis, Category 2/3. In his narrative report, he related an occupational history of ten years of underground coal mining, and a smoking history, commencing in the miner's teens and ceasing in 1989, of 2 packs a day.[2] He was of the view that the miner's conditions were caused by a previous, severe, cigarette habit, and not his employment. Noting no prior history of treatment for a pulmonary condition before his open heart surgery, Dr. Seargent concluded that the miner's disability was due to his cardiac condition, and not chronic obstructive pulmonary disease or pulmonary fibrosis. (DX 11).

Dr. Steve Kraman found the Claimant was not disabled due to a chronic pulmonary impairment
related to his coal mine employment. [3] (DX 26, DX 10)

Dr. Lane reported that Claimant's x-rays were interpreted as demonstrating simple coal workers' pneumoconiosis. He diagnosed severe obstructive airways disease. Dr. Lane's opinion does not directly address the issue of total disability and its cause. Instead, he focuses on whether the miner's chronic obstructive pulmonary disease is due to coal dust or to smoking. While the thrust of the doctor's opinion seeks to rebut Dr. Parrish's opinion that coal workers' pneumoconiosis played a role in the development of the miner's obstructive lung disease, his opinion is nonetheless considered since he found a severe degree of obstructive airways disease. Because a severe chronic airways obstruction may cause a disabling respiratory impairment, the doctor's opinion relating to the cause of that impairment is relevant to a determination relation to disability causation..

Dr. Lane diagnosed coal workers' pneumoconiosis and severe airways disease. He disagreed with Dr. Parrish' opinion that smoking as well as coal workers pneumoconiosis, contributed to the

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[2] The Claimant was born in October 8, 1942. (DX 11) At the hearing, he testified he started smoking about age 18 or 19 and stopped in 1989 (Tr. 14), at age 47. Claimant essentially denied smoking three packs a day for the 29 years. He indicated he started out at less than a pack, and that his habit increased over time. He estimated that he smoked at most about two packs a day in later years. I find his testimony credible. However, based on the evidence, I also find it difficult to quantify the extent of his smoking habit over the 29 years he admits he smoked. Nonetheless, the physicians general assessment of the extent of the Claimant's smoking history as "exceedingly significant" or "very heavy" is a fair characterization.

[3] In my prior decision, I accorded Dr. Kraman's report little weight because he merely checked a box indicating total disability was unrelated to coal mine employment, without explanation. The Board in its decision remanding this case for further consideration upheld that determination. *Director, OWCP v. Rowe,* 710 F. 2d 251, 255 , 5 BLR 2-99, (6TH Cir. 1983)

miner's obstructive pulmonary impairment. He was of the opinion that simple coal workers' pneumoconiosis (i.e. clinical pneumoconiosis) [4] does not cause an obstructive airways disease. He dismissed almost entirely the general principle that coal dust can cause an obstructive impairment. He ruled out coal dust as a cause of COPD, under the broad assumption that the most common cause is cigarette smoking. He formed this conclusion relying on the general premises that: (1) centrilobular emphysema in persons exposed to large concentrations of coal mine dust is due to coal dust in less than 5% of the cases; and (2) simple coal workers' pneumoconiosis does not cause an obstructive impairment. (DX 37) While Dr. Lane recognized that emphysema is seen in a small number of cases, where persons are exposed to large concentrations of coal dust, he ruled out coal dust related emphysema in this case without explanation.

Dr. Broudy did not find sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis. He diagnosed severe COPD, with some secondary restriction of vital capacity, most likely due to cigarette smoking, noting that cigarette smoking causes obstructive impairment by way of chronic obstructive bronchitis and/or pulmonary emphysema. He stated that the restrictive component noted in Claimant's medical records was due to severe obstruction. Dr. Broudy did not believe that simple coal workers' pneumoconiosis could cause pulmonary disability in the absence of complicated pneumoconiosis and a restrictive impairment. He ruled out coal workers' pneumoconiosis as a cause of the pulmonary impairment, finding neither complicated coal workers' pneumoconiosis nor a restrictive defect. He found Mr. Baird's ventilatory defect to be obstructive, noting that the restrictive component was not due to some other independent cause. He concluded there was no significant pulmonary disease or respiratory impairment which arose from the inhalation of coal dust. The Claimant in his view would have suffered from obstructive airways disease due to cigarette smoking whether or not he worked in the coal mines. (DX 37, EX 4).

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[4] Sixth Circuit in *Cornett v Benham Coal Co.*, 227 F. 3d 569 (2000) citing *Kline v. Director, OWCP,* 877 F. 2d 1175, 1178 (3d Dir. 1989) and *Hobbs v. Clinchfield Coal Co.*, 45 F. 3d 819, 821 (4th Cir. 1995) has recognized that the "legal" definition of pneumoconiosis encompasses a wider range of afflictions than does the more restrictive medical definition of pneumoconiosis. Medical pneumoconiosis is a particular disease of the lung generally characterized by certain opacities appearing on a chest x-ray. *See Usery v. Turner Elkhorn Mining Co.,* 428 U.S. 1, 6-7, 96 S.Ct. 2882, 49 L.Ed.2d 752 (1976); *see also Hobbs,* 45 F.3d at 821 Clinically, pneumoconiosis may be described in simple terms as a chronic lung disease marked by the fibrotic reaction of lung tissue to inhaled coal dust. Legal pneumoconiosis is a much broader category of diseases, which includes but is not limited to medical, or "coal workers'," pneumoconiosis. "Legal pneumoconiosis" includes "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment". *See Clinchfield Coal Co. v. Fuller,* 180 F.3d 622, 625; *Hobbs,* 45 F.3d at 821; *see also* 20 C.F.R. §§ 718.201 A "medical diagnosis finding no coal workers' pneumoconiosis is not equivalent to a legal finding of no pneumoconiosis." *Hobbs,* 45 F.3d 821. *Island Creek Coal Co. v. Compton,* 211 F. 3d 203 (4th Cir. 2000).

Dr. Dahhan examined the miner on two occasions and evaluated his pulmonary condition. He diagnosed simple coal workers' pneumoconiosis, Category II, and chronic obstructive pulmonary disease (chronic bronchitis and emphysema). He found a totally disabling obstructive ventilatory defect with partial reversibility due to smoking. He found the miner's COPD (chronic bronchitis and emphysema) to be unrelated to coal dust or simple coal workers' pneumoconiosis. His reasons were: (1) there was no evidence of complicated pneumoconiosis which can cause an obstructive abnormality. (2) there was no evidence of a restrictive ventilatory impairment; (3) the miner had not had exposure to coal dust since 1991, a period sufficient to cause cessation of any industrial bronchitis. (4) the miner's obstructive defect showed significant reversibility with administration of bronchodilators, a finding he believed to be inconsistent with the permanent effects of coal dust on the respiratory system; and finally he opined that (5) coal dust does not cause the development of severe obstructive abnormality, citing studies authored by Lapp, Morgan, Zaldivar, "Airways Obstruction, Coal Mining and Disability", Occupational and Environmental Medicine, 1994; 51:234-238; and by Thurlbeck, "Pathophysiology of COPD", Clinics in Chest Medicine, Vol. II, No. 3, 1990. Dr. Dahhan's opinions were formed, in part, based on empirical studies which examined the question of whether inhalation of coal dust in the absence of complicated coal workers' pneumoconiosis or smoking can lead to disabling airways obstruction. The study concluded that if so, it was a rare occurrence. The study focused on the frequency that significant airways obstruction is found in non-smoking miners with progressive massive fibrosis (i.e. complicated coal workers' pneumoconiosis). (EX 7, EX 5, DX 56 and DX 67)

Dr. Fino diagnosed coal workers' pneumoconiosis by chest x-ray, and an obstructive lung abnormality consistent with centrilobular emphysema caused by smoking. He believed the miner's had a disabling impairment due to smoking. He found no significant pulmonary fibrosis due to coal workers' pneumoconiosis because the miner did not, in his view, have a restrictive impairment, or a drop in pO2. Dr. Fino did find a reduction in diffusing capacity but believed it to be reflective of anatomic emphysema secondary to smoking, and not coal mine dust inhalation. (DX 71)

Dr. Morgan reviewed the miner's medical records, but in the absence of serial x-rays before and after the miner's coronary surgery, he did not find the x-ray evidence sufficient to justify a diagnosis of coal workers' pneumoconiosis. He also could not reach a conclusion as to the exact cause of the x-ray changes. He stated that they could be due to simple coal workers' pneumoconiosis, silicosis, or sarcoidosis.

Dr. Morgan reviewed the pulmonary functions tests over time, 1991 to 1997, and concluded that the miner has a significant disabling respiratory impairment, i.e., a mild to moderate obstruction and a significant restrictive impairment. In 1991, he stated that the pulmonary function test results showed a mild obstruction. He believed the reduction in FEV_1 and FVC was mainly the consequence of Claimant's restrictive impairment resulting from his coronary artery by-pass surgery. Dr. Morgan believed that while there may have been a "deterioration in Claimant's lung volumes due to increasing

restriction, the extent of the restriction was appreciably less than the concomitant decline in lung function caused by obstruction". As a result, his FEV_1/FVC declined significantly between 1991 and 1997, when he was no longer exposed to coal dust.

While he found the Claimant smoked enough cigarettes for his obstructive airways disease to be severe, he was not sure he would agree with Dr. Broudy's conclusion that the restrictive component was due to the severe obstruction. Further, he could not explain why it has become progressively worse since he stopped smoking in 1989. (DX 72)

Dr. Branscomb did not find enough evidence to justify a diagnosis of CWP, noting that simple coal workers' pneumoconiosis does not progress once exposure to coal dust ceases and that there was no x-ray evidence of coal workers' pneumoconiosis prior to 1991(EX 2. p. 26). He stated that, if heart disease were not considered, findings could erroneously be attributed to dust exposure (EX 2 p. 46). However, he dismissed coal dust as a contributing factor, indicating that the progression of changes would not be consistent with coal workers' pneumoconiosis since there is no data to indicate that once a person leaves the mines, he can later develop positive findings of pneumoconiosis, if they were not present at the time he left. (Ex 2, p. 49-50). Dr. Branscomb believed the miner's heart disease explains his clinical findings and that cardiovascular disease is a much more likely explanation for the changes noted on the Claimant's x-ray. He found a totally disabling respiratory impairment, obstructive and not restrictive, caused by smoking. He concluded that , assuming the miner has coal workers' pneumoconiosis, his pulmonary disability is neither caused or aggravated in whole or part by coal dust inhalation.. (EX 1, EX 2 p. 38-40)

Dr. Repsher reported that the chest x-rays were positive for simple coal workers' pneumoconiosis but not complicated pneumoconiosis. He indicated that the pulmonary function studies document slowly progressive, very severe chronic obstructive pulmonary disease with an impairment of diffusing capacity and no evidence of restrictive lung disease. He believed this clinical pattern to be characteristic of cigarette smoke induced chronic obstructive pulmonary disease with emphysema. Dr. Respher believed it possible but unlikely for simple coal workers' or coal dust exposure to cause a disabling obstructive respiratory impairment. He indicated that simple coal workers' pneumoconiosis does not cause a measurable pulmonary impairment and does not add any detectable additional impairment other than one would expect from cigarette smoke induced COPD. He based his opinion on the assumptions that: (1) coal miners with simple coal workers' pneumoconiosis have normal pulmonary function; and (2) coal workers' pneumoconiosis when clinically significant is primarily a restrictive disease that may have some superimposed obstructive features. Finally, he attributed the progression in the miner's impairment to the aging process since the miner had stopped smoking, and to his heart disease. (EX3, EX 6, EX 9). He was of the opinion that the miner's severe pulmonary respiratory impairment was not related to pneumoconiosis, but rather to cigarette smoke induced COPD and emphysema. He thus concluded that the miner's disability was due to cigarette smoke induced COPD and emphysema, and his cigarette smoke aggravated coronary artery disease.

DISCUSSION

A miner who has pneumoconiosis is totally disabled "if the impairment prevents him from engaging in gainful employment requiring the skills and abilities comparable to those of any employment iin the mine or mines in which he previously engaged with some regularity and over a substantial period of time". 30 U.S.C. 902(f)(1)(A). In the Sixth Circuit, the miner must prove by a preponderance of the evidence that his total disability is "due at least in part" to pneumoconiosis under *Adams v. Director, OWCP*, 886 F. 2d 818, 825 (6th Cir. 1989), and that pneumoconiosis is more than a *de minimus* or "infinitesimal" contribution to disability. *See. Peabody Coal Co. v. Smith*, 127 F. 3d 504, 507 (6th Cir. 1997). The amended regulations define "disability due to pneumoconiosis"as "a substantially contributing cause" of the miner's disability to harmonize the various interpretations in the circuit court decision . While the Sixth Circuit has indicated that the standard in *Adams* places a lesser burden on Claimant than the "substantially contributing cause" standard articulated in the Third, Fifth and Eleventh Circuits, the Department found that the standards developed by the Courts of Appeals since 1989 varied little. The Department further recognized that the "due at least in part" standard is encompassed within and satisfies the "substantially contributing cause" standard articulated in revised regulation *20 C.F.R. 718.204*. Thus, a diagnosis of pneumoconiosis, emphysema and chronic obstructive lung disease is sufficient to satisfy the definition of disability due to pneumoconiosis under the new regulations. *Adams*, 886 F. 2d 818, 826. *65 Fed Reg. 79947*

In evaluating the evidence under Section 718.204, the Department of Labor's ("DOL") comments in the revised regulations are instructive on issues concerning the relationship of coal mine dust to chronic obstructive pulmonary disease and respiratory disability arising out of obstructive airways diseases. DOL's position is that the substantial weight of the medical scientific literature documents that, in addition to the risk of simple coal workers pneumoconiosis and pulmonary massive fibrosis, coal miners have increased risk of development of chronic obstructive pulmonary disease. See NIOSH *Criteria for a Recommended Standard, Occupational Exposure to Respirable Coal Mine Dust*(1995), ("NIOSH Criteria") *62 Fed. Reg. 3343* (January. 22, 1997); *65 Fed. Reg. 79939* (December 20, 2000). The Department rejected the notion that clinically significant obstruction resulting from coal dust inhalation occurs only in cases of severe fibrosis where a combined obstructive and restrictive defect is present. Rather, it found that coal dust related obstructive lung diseases may cause clinically significant reduction in lung function independent of coal workers' pneumoconiosis. In fact, lung function measured as FEV_1 has been shown both in cross sectional and longitudinal studies to decline in relation to underground coal dust exposure. The decline occurs in similar rates in smokers and non smokers although overall loss of lung function is greater in smokers, the two effects being cumulative. See, Seaton in "Coal Workers Pneumoconiosis", in Morgan WKC, Seaton A., eds. *Occupational Lung Diseases* (WB Saunders Co., 3d ed. 1995) 374-406, 65 Fed. Reg. 79939. Further, scientific studies show that a clear relationship exists between dust exposure and a decline in pulmonary function, even in miners with no radiographic evidence of clinical coal workers'

pneumoconiosis. Attfied MD, Hodus, TK "Pulmonary function of U.S. coal miners related to dust exposure estimates". *Am Rev. Respir Dis*. 145:605-609 (1992). In evaluating the weight to be accorded to the various physician opinions, I make the following findings.

All the physicians, except Dr. Branscomb, unequivocally diagnosed simple coal workers pneumoconiosis. They all agree that the miner has a disabling obstructive impairment. All, except Drs. Parrish, Dr. Seargeant, and Dr. Morgan agree that the miner's disabling pulmonary/ respiratory impairment is due to cigarette smoking. While Drs. Lane, Broudy, Fino, Dahhan and Branscomb provide slightly different rationales for their opinions, none of these physicians believe that coal dust is a contributing factor in the miner's disability. In their view, simple coal workers' pneumoconiosis does not cause an obstructive pulmonary impairment. Dr. Respher essentially agrees with Drs. Lane, Broudy, Fino Dahhan, and Branscomb. He is of the view that simple coal workers' pneumoconiosis causes an insignificant airways obstruction, and that coal miners with the disease have normal pulmonary function. Drs. Morgan and Seargent believe that the miner's disabling pulmonary impairment is due to cardiac disease. Dr. Parrish believes that his disabling impairment is related, both to simple coal workers' pneumoconiosis, and obstructive lung disease caused by coal mine employment and cigarette smoking. To the extent that the opinions of Drs. Broudy, Branscomb, Dahhan, Fino, and Respher are based on assumptions and conclusions which are inconsistent with the scientific findings discussed herein and adopted by the Department of Labor in the Comments to the amended regulations, the probative value of their opinions is significantly diminished.

For example, Dr. Dahhan ruled out coal dust as a cause of the miner's severe obstructive abnormality based on a scientific article which reached conclusions contrary to the prevailing view in the medical community. He eliminated both "legal" and simple "clinical" coal workers' pneumoconiosis as a contributing factor to the miner's obstructive airways disease, by relying on the premise that only complicated coal workers' pneumoconiosis can cause an obstructive impairment. As discussed above, the substantial weight of the medical and scientific studies refutes this premise. Secondly, he rejected the concept that the miner's chronic obstructive disease could be related to coal dust, since the miner had not been exposed to coal mine dust since 1991. Thus, he did not take into account that the cumulative effect of coal dust in the lung dysfunction of miners who were also smokers, or the progressive effect of such a coal dust related obstructive pulmonary dysfunction. *See Orange v. Island Creek Coal Co., 786 F. 2d 724, 727 (6th Cir. 1986)*. See also Coggon & Newman-Taylor, 1998, "*Coal mining and obstructive pulmonary disease: a review of the evidence": British Coal Respiratory Disease Litigation*"; Donnan et. al, 1997, "*Progress of simple pneumoconiosis in ex-coal miners after cessation of exposure to coal mine dust*." Finally, and most significantly, he based his opinion on the underlying reasoning that coal dust does not cause the development of a severe obstructive abnormality. As noted above, this latter rationale was reached, in part, based on scientific studies which examined the question of whether inhalation of coal dust in the absence of complicated coal workers' pneumoconiosis or smoking can lead to disabling airways obstruction. The findings and conclusions reached in that study have been refuted by scientific studies, generally accepted in the medical

community, which indicate that coal mine dust can cause a clinically significant obstructive abnormality. See Comments, even in the absence of "clinical pneumoconiosis. 65 Fed. Reg. 79940-79942 (December 20, 2000).

Dr. Broudy's opinion that the Claimant's obstructive impairment is not related to coal dust inhalation is also problematic. The focus of his opinion was on "clinical" coal workers' pneumoconiosis (and only then on complicated pneumoconiosis as a source of disabling pulmonary impairment). He appears to reject the statutory presumption that simple coal workers' pneumoconiosis can cause pulmonary disability. Such an opinion is contrary to prevailing case law and the Statute. Further, Dr. Broudy's opinion that the miner would have suffered from COPD whether or not he worked in the coal mines begs the question of whether coal dust contributed in any substantial measure to his respiratory disability. The doctor ruled out coal dust exposure as a causative factor in the miner's pulmonary disability under the assumption that "clinical" pneumoconiosis when it causes an impairment is usually in complicated form and creates a restrictive defect. He did not consider whether any portion of the claimant's pulmonary disability was related to coal dust induced COPD. Nor did he take into account the additive effect of coal dust where COPD was due to cigarette smoking. The medical data indicates that smokers who mine have additive risk of developing significant obstruction. "Even in the absence of smoking, coal mine dust exposure is clearly associated with clinically significant airways obstruction and chronic bronchitis. The risk is additive with cigarette smoking." *See, 65 Fed Reg 79940*. *See also,* Attfield and Hodous, "Pulmonary function of U.S. Coal miners related to dust exposure estimates," Am Rev Respir Dis 145:605, 609 (1992); *NIOSH Criteria* §4.2.2, Rulemaking Record, Exhibit 2-1 at 51 (there exists a clear relationship between dust exposure and a decline in pulmonary function, even in miners with no radiographic evidence of clinical coal workers' pneumoconiosis.) Thus, I find Dr. Broudy's opinion on the cause of the miner's disabling respiratory impairment to be substantially undermined and of very little probative value.

Dr. Respher's opinion is not substantially different from that of Drs. Dahhan and Broudy in that he believes that simple pneumoconiosis causes an insignificant airways obstruction and that coal miners with simple coal workers pneumoconiosis does not cause clinically significant or potentially disabling obstructive impairment. As his opinion is based on an erroneous assumption, it is not credited for reasons similar to those discussed *supra* in the evaluation of Dr. Dahhan's opinion.

Dr. Branscomb found an obstructive impairment of moderate and possibly severe degree. He attributed the obstructive impairment to smoking because there was no evidence of complicated pneumoconiosis which would create an obstruction, and because the lung volume tests consistently showed no restrictive change, but rather features of obstruction such as asthma or COPD, which are incompatible with coal workers' pneumoconiosis. Finally, Dr. Branscomb indicated that he could not exclude coal workers' pneumoconiosis, but found the evidence insufficient to justify a diagnosis. In his view, if the x-rays were negative when the miner left the mines, he could not develop positive findings thereafter.

Dr. Branscomb's opinion is not credited for several reasons. First, his opinion focuses only on whether there was sufficient evidence to justify a diagnosis of "clinical" coal workers' pneumoconiosis, and not whether the disease, in the broader definition, is a causal factor in the miner's disability. Secondly, his opinion is at variance with the generally accepted medical finding that chronic obstructive pulmonary disease includes three disease processes characterized by airways dysfunction (i.e., chronic bronchitis, emphysema, and asthma) which may be caused by or aggravated by coal mine dust. To the extent that Dr. Branscomb's opinion excludes these diseases from the definition of coal workers' pneumoconiosis, his opinion is rejected. *See Hughes v. Clinchfield Coal Co.* 21, BLR 1-134. Thirdly, he rejected the medical studies which indicate that dust exposure without "clinical" pneumoconiosis can result in airways obstruction, and I do not find his effort to distinguish Mr. Baird from the persons in those studies to be very persuasive. To the extent that he agrees with Dr. Dahhan, his opinion is not credited for the same reasons Dr. Dahhan's opinion on this point was not accepted. See 65 Fed Reg. 79940-79942 (December 20, 2000)

Further, Dr. Branscomb's opinion is given very little probative weight as it is based on the erroneous assumption that because the miner did not have coal workers' pneumoconiosis when he left the coal mines, pneumoconiosis could not have progressed once exposure to coal dust ceased. Thus, he excluded coal dust as a factor in the miner's disabling impairment, and attributed his impairment to heart disease instead. This assumption is inconsistent with the general principle that coal workers' pneumoconiosis is an irreversible progressive disease which may manifest itself after coal dust exposure ceases. *See Orange v. Island Creek Coal Co., 786 F. 2d 724, 727 (6th Cir. 1986)*. See also Coggon & Newman-Taylor, 1998, "*Coal mining and obstructive pulmonary disease: a review of the evidence": British Coal Respiratory Disease Litigation*"; Donnan et. al, 1997, "*Progress of simple pneumoconiosis in ex-coal miners after cessation of exposure to coal mine dust*"; "*Adverse effect of crystalline silica exposure*" American Thoracic Society (ATS) 1997. *64 Fed. Reg. 54979* (October 8, 1999) and *62 Fed. Reg. 3337, 3344. See also* P. Francois, et al, "Pneumoconiosis of Delayed Apparition: Large Scale Screening in Population of Retired Coal Miners of North Coal Fields of France," in Seventh Annual Pneumoconiosis Conference, Abstracts of Communication 979 (1988).

Moreover, while there are no medical records in this file that pre-date the miner's quadruple bypass surgery which include a diagnosis of pneumoconiosis, the doctor's assumption that the miner did not have the disease when he left the mines is no more than speculation. His opinion is not based on objective medical evidence, and there is no conclusive evidence that pneumoconiosis did not exist. Dr. Williams, the cardiovascular surgeon who performed the miner's coronary artery by-pass, noted the presence of moderate to severe anthracosis in the lungs during his surgical procedure. While Dr. Williams was not certain whether coal dust explained his finding, Dr. Clark's diagnosis of the disease by positive chest x-ray in September of 1991, some three months after the Claimant last worked in the mines, was sufficiently close in time to the miner's last coal mine employment to give rise to an inference of the existence of the disease at the time he left the mines.(DX 36)

Dr. Morgan did not find sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis. It would appear that, in light of what he viewed to be inconclusive evidence of coal workers' pneumoconiosis, he concluded that the miner's continued decline in lung function after he ceased his employment in the mines was due only to his coronary artery disease. He believed the miner's cigarette smoking was extensive enough to result in obstructive airways disease, but he could not account for why the condition became progressively worse after he stopped smoking in 1989..

He concluded the miner's restrictive airways disease was due to heart disease and his obstructive disease was due to smoking. Dr. Morgan's opinion regarding the cause of Claimant's obstructive impairment and its contribution, or lack thereof, to his respiratory disability lacks a sufficient rationale. He focuses primarily on the cause of the miner's restrictive impairment, and does not state clear reasons why he discounted the miner's coal mine employment as a causal factor in his obstructive impairment. Given his ambivalence relating to the issue of whether the miner has pneumoconiosis, and in light of his inability to attribute a cause to the progressive increase in the miner's obstructive lung disease after he ceased smoking, Dr. Morgan's opinion on disability causation is not fully credited. [5]

> Dr. Sargeant reached his opinion based, in large part, on the fact that there are no records to show that Claimant was treated for a pulmonary condition until after his heart surgery. Dr. Sargeant's opinion on the issue of cause of disability is not very persuasive, because he does not explain his reasons for excluding Claimant's fifteen years of coal mine exposure as a contributing factor to his disability or state findings based on objective medical data or tests to support his opinion that the miner's cardiac condition is the cause of his disability and not COPD or pulmonary fibrosis. He indicated that he had considered all the Claimant's medical records he could gather, but he makes no reference to objective medical data upon which he relied in reaching his opinion. Dr. Sargeant's opinion is not well reasoned. On one hand he makes a clinical finding of moderately severe coal workers' pneumoconiosis in 1994, but on the other completely discounts the significance of that finding in reaching his ultimate conclusion on the cause of the miner's disability. Since there are no medical records in the record which pre-date the miner's quadruple bypass

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[5] A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. *See Hoffman v. B&G Construction Co.*, 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984). A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. *Fields, supra*. Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder-of-fact to decide. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989)(en banc). Moreover, statutory pneumoconiosis is established by well-reasoned) medical reports which support a finding that the miner's pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP*, 11 B.L.R. 1-135 (1988).

surgery, there is no independent bases to support the doctor's emphasis on the absence of any evidence of the disease prior to the miner's heart surgery. There likewise is no independent bases to determine that it did not exist at the time he left the miners and progressed to the stage of manifesting itself thereafter. Further, although he may have considered the medical records relating the miner's heart surgery, he does not address the significance, if any, of: (1) the observations of Dr. Williams, the cardiovascular surgeon who performed the miner's coronary artery bypass, that there was evidence of severe anthracosis in the lungs; or (2) the findings of Dr. Clark in her pulmonary evaluation post surgery of severe pneumoconiosis based on chest x-ray. I do not consider his opinion to be well documented, and I do not accord his assessment of disability causation great probative weight.

Dr. Lane ruled out coal mine dust as a contributing factor in the miner's disabling respiratory impairment, based on the assumption that "clinical" (simple) coal workers pneumoconiosis does not cause airways obstruction. However, I do not find Dr. Lane's opinion to be well reasoned. It is not evident from this record what medical evidence he considered in reaching his conclusions that the miner's simple coal workers' pneumoconiosis does not cause an obstructive pulmonary disability or that his disabling obstructive airways disease is not, in part, caused by coal dust exposure. The broad, general reasons which he states as a bases for finding that coal dust is not related to this Claimant's disabling respiratory impairment do not satisfy the requirements for a well reasoned and documented opinion. I, therefore, accord his opinion less weight.

Finally, Dr. Fino ruled out coal dust as a factor in the miner's respiratory disability under the theory that pulmonary fibrosis due to coal workers' pneumoconiosis, if significant, would result in a restrictive defect. He found no evidence of a restrictive defect, and thus, no basis to conclude that coal dust played a factor in the miner's lung dysfunction. Rather he believed that centrilobular emphysema caused by smoking accounted for the obstructive abnormality. Dr. Fino does not discuss the findings upon which he based his conclusions. However, his response to the proposed revised regulations, made during the comment period about the same time he rendered his opinion herein, sheds considerable light on his thinking on respiratory disability causation in coal miners. Dr. Fino adhered to the position that there is no evidence of a clinically significant reduction in lung function resulting from coal dust exposure. (Rule Making Record, Exhibit 89-37, Appendix C) 65 Fed. Reg. 79938-79939. Specifically, he stated that "[W]hile there is no doubt that come miners have clinically significant obstruction as a result of coal mine dust inhalation, it occurs in cases of severe fibrosis where a combined obstructive and restrictive defect is present. ... [T]here is no evidence that there is a clinically significant reduction in the FEV_1 as a result of chronic obstructive lung disease due to coal mine dust inhalation." Dr. Fino, cited with approval the findings of Dr. Morgan

two decades previously, and stated that while coal mine dust may cause slight clinically insignificant decreases in the FEV_1 in some miners, there is no evidence that these decreases cause or contribute to pulmonary disability or causes or contributes to obstructive lung disease. In a separate comment to the revised regulations, Dr. Fino in his review of relevant medical studies recognized that the amount of emphysema in the lungs of miners increases with the severity of simple coal workers pneumoconiosis, but found no evidence of a clinically significant deterioration in lung function as the emphysema worsens. Rule Making Record, Exhibit 89-37, Appendix C, at 32-33 (65 F.R. 79939 10/20/2000).

The Department of Labor, after review of all the medical and scientific data, rejected Dr. Fino's positions as not in accord with prevailing view in medical community or the substantial weight of medical and scientific literature. Based on those findings which are adopted herein, Dr. Fino's opinion is rejected. Specifically, clinical studies and scientific evidence regarding cellular mechanisms of the lung injury link in a substantial way, coal mine dust exposure to pulmonary impairment and chronic obstructive lung disease. DOL has unequivocally found the scientific data sufficient to document that coal mine dust can cause chronic obstructive pulmonary disease which includes chronic bronchitis and emphysema, and associated airways obstruction. NIOSH concluded that "[i]n addition to the risk of simple CWP and PMF (progressive massive fibrosis), epidemiological studies have shown that coal miners have an increased risk of developing COPD." The Department adopted the views that: "coal mine dust can cause chronic airflow limitation in life and emphysema at autopsy, and that this may occur independently of CWP." It adheres to the position that there exists "a statistically significant association between cumulative dust exposure and decline in lung function, and that coal mine dust can be the cause of chronic bronchitis." *65 Fed Reg. 79939.* (Citations omitted). This position is based, in part, on Marine's cross-sectional 1988 study of coal miners which found clinically significant decreases in pulmonary function in both smokers and non-smokers. Marine WM, et al., "Clinically important respiratory effects of dust exposure and smoking in British coal miners". Am Rev Resp Dis, 137:107-112 (19888) see also *NIOSH Criteria* §4.2.2.1, Rulemaking Record, Exhibit 2-1 at 52. Marine's findings showed that the" incidence of non-smoking coal miners with intermediate dust exposure developing moderate obstruction (FEV_1 of less than 80%) is roughly equal to the incidence of moderate obstruction in smokers with no mining exposure". Similarly, the incidence of non-smoking miners with intermediate exposure developing severe airways obstruction (FEV_1 of less that 65%) is equal to the incidence of severe obstruction in non-mining smokers." Thus, smokers who mine have additive risk of developing significant obstruction. Thus, "[e]ven in the absence of smoking, coal mine dust exposure is clearly associated with clinically significant airways obstruction and chronic bronchitis. The risk is additive with cigarette smoking." *See, 65 Fed Reg 79940. See also*, Attfield and Hodous, "Pulmonary function of U.S. Coal miners related to dust exposure estimates," Am Rev Respir Dis 145:605, 609 (1992); *NIOSH Criteria* §4.2.2, Rulemaking Record, Exhibit 2-1 at 51 (there exists a clear relationship between dust exposure and a decline in pulmonary function, even in miners with no radiographic evidence of clinical coal workers' pneumoconiosis.)

Upon consideration of all the physician opinions and the relevant medical evidence, I find Dr. Parrish's opinion regarding the cause of Claimant's totally disabling respiratory impairment to be the most probative. Dr. Parrish is a qualified pulmonary specialist. He has treated the Claimant for over six years for his respiratory impairment and coal workers' pneumoconiosis. Although the revised regulations apply only to evidence developed after January 19, 2001, the provisions of Section 718.104 codify judicial precedent and are instructive in determining the weight to be accorded the opinion of the treating physician. As Claimant's treating physician, I find Dr. Parrish's opinion entitled to great weight. *Tussey v. Island Creek Coal*, 982 F. 2d 1036, 17 BLR 2-16 (6th 1993). *Cf Griffitih v. Director, OWCP* 49 F. 3d 184 19 BLR 2-11 (6th Cir. 1995). His treatment notes document that he has regularly examined the miner on follow up visits, monitored his condition by diagnostic tests (pulmonary function studies and chest x-rays), and prescribed a course of medication therapy. His opinion is documented by the objective medical evidence and satisfies the requirement for credible evidence. His opinion is further consistent with the underlying purposes of the Act, in that it is incorporates the broad definition of coal workers pneumconiosis, and recognizes the irreversible progressive nature of the disease process. Dr. Parrish attributes the miner's disability to the risk factors of smoking and coal dust exposure. While he cannot definitively state the degree to which either factor contributes to the miner's respiratory disability, his inability to do so does not affect the weight which can be given to his opinion. The courts have recognized that the question of the relative amounts that various causal elements contribute to a totally disabling respiratory impairment can be extremely problematic. *See Adams*, 886 F. 2d 825; *Cross Mountain Coal Co. v. Ward*, 93 F. 3d 211, 218 (6th 1996); *Compton v. Inland Steel Coal Co.*, 933 F. 2d 477, 481- 483 (7th Cir. 1991).

Therefore, based on the foreging, I find that Claimant has established that his total disability is due in part to pneumoconisis. As such, he is entitled to benefits under the Act.

ORDER

Key Mining, Inc., Kline Coal Company, and their insurer, American Mining Insurance Company, are hereby ORDERED to pay the following:

(1) To Claimant, Charles E. Baird, all benefits to which he is entitled under the Act, augmented by reason of one dependent, commencing June W, 1994; and

(2) To Claimant, all medical and hospitalization benefits to which he is entitled, commencing June 1, 1994.

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MOLLIE W. NEAL
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this Decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this Notice of Appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor, 200 Constitution Avenue, N.W., Room N-2117, Washington, D.C. 20210.